CORONARY OCCLUSION—SOME ABNORMAL RHYTHMS*

REVIEW OF LITERATURE

REPORT OF CASES

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DISCUSSION *by Donald J. Frick, M. D., Los Angeles; William Dock, M. D., San Francisco; V. R. Mason, M. D., Los Angeles.*

LEWIS [1] has shown that ligation of a coronary artery in animals may provoke disturbances in rhythm. In man extrasystoles are common, and paroxysmal tachycardia, especially of the ventricular type, auricular flutter, auricular fibrillation, and heart block have all been observed. Robinson and Hermann [2] have commented on the frequency of ventricular tachycardia following coronary occlusion. Wearn [3] reported a study of nineteen cases of coronary infarction which came to autopsy. There were ten electrocardiograms in this series. It was noted that disturbances in the T wave and a decrease in the amplitude of QRS were the most constant findings. In all the ten records there was an alteration of T in at least one lead, particularly in 1 and 2. There were no abnormal rhythms reported.

Fred M. Smith [4] reported eleven patients with coronary occlusion, in one of whom the descending branch of the left coronary artery was ligated in repairing a stab wound of the heart; the remaining ten patients had typical clinical manifestations of coronary artery occlusion. There were two autopsies in this series. The most constant findings were change in T deflection and a decrease in amplitude of QRS. No abnormal rhythms were described.

K. Shirley Smith [5] reports one case of complete heart block following coronary thrombosis. The patient recovered. Levine and Brown [6] found only two instances of complete heart block in a series of 145 cases and Parkinson and Bedford [7] only one in a series of 100 cases of cardiac infarction; abnormal rhythms other than extrasystoles occurred in fourteen. These authors are of the opinion that in the majority of cases the rhythm remains normal apart from extrasystoles, and the rate is usually but not always increased.

Louis H. Sigler [8] reported a series of twenty cases of acute coronary occlusion. These patients were apparently studied only from the clinical and electrocardiographic standpoint as no autopsies were reported. Except in one patient whose electrocardiogram showed auricular fibrillation no abnormal rhythms were demonstrated. There were two records which showed partial heart block. Many of the electrocardiograms in this series were taken several weeks or months after the attack, therefore no conclusions can be reached concerning disturbances in rhythm or conduction.

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FINDINGS IN EXPERIMENTAL STUDY OF CORONARY INTERFERENCE

In a recent study of angina pectoris F. C. Wood and C. C. Wolferth [9] demonstrated temporary ventricular complex changes in fifteen patients during attacks of angina pectoris. The remaining fifteen showed no "specific" electrocardiographic changes during the attacks. In a series on dogs and cats, temporary interference with a part of the coronary circulation produced temporary and rapidly reversible changes in the electrocardiogram somewhat analogous to those seen during attacks of angina pectoris. More striking changes followed the clamping of the vessels on the posterior surface of the heart (the circumflex branch of the left and the posterior descending branch). Usually little or no change was produced by obstruction of the right coronary or of the anterior descending branch of the left coronary artery. There were more marked changes which followed the simultaneous obstruction of both main branches of the left coronary artery than followed the obstruction of either one separately. In their experiments it was also noted that rapidly reversible alterations in the ventricular complexes were more readily obtained after myocardial damage, incident to previous manipulation, had produced a certain amount of permanent electrocardiographic change. This would indicate that coronary occlusion in a healthy fresh heart does not produce electrocardiographic changes as readily as it does in a damaged one. Wood and Wolferth noted further that temporary coronary occlusion in their animals frequently produced no electrocardiographic change. Cardiac arrhythmia, which could be attributed to the circulatory disturbance in itself, was not a frequent early phenomenon. When it did occur, it seemed to be attributable to the mechanical stimulation of the heart muscle by the mechanism producing the occlusion.

STUDY OF THE REPORT OF EIGHTEEN CASES OF CORONARY OCCLUSION

This paper is based on a study of eighteen cases of coronary occlusion in which necropsy revealed obstruction by a thrombus or an embolus (one case) in some portion of the coronary circulation. Electrocardiograms were obtained in each case following the occlusion; the shortest period elapsing was four hours, the longest twenty-one days. In eight cases the first electrocardiogram was taken within thirty-six hours, in six within one week, and in four the records were not obtained until more than seven days had elapsed following the occlusion. More than one electrocardiogram was obtained in several cases.

Age Incidence and Etiology.—The youngest patient in this series was forty-three and the oldest seventy-seven, the average age being fifty-nine. The proportion of males was considerably higher than is usually given, as there were sixteen males and only two females. As far as could be determined from the histories—some of which through force of circumstances were very sketchy—occupation, habits and previous illnesses

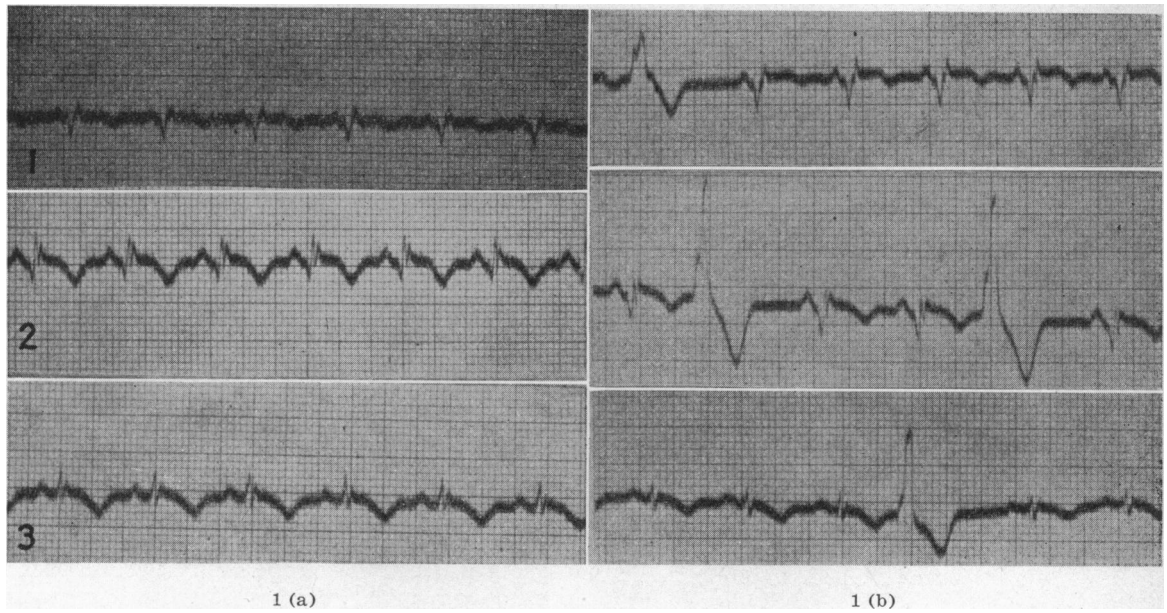

Fig. 1 (a) is an electrocardiogram taken March 31, 1927, showing low voltage in all leads and a high take-off of R-T in leads 2 and 3. This record was considered typical of coronary occlusion. Figs. 1 (b), 1 (c), and 1 (d) are electrocardiograms taken during the subsequent five months.

could not be held responsible for the coronary artery disease. The Wassermann test was positive in one case and a two plus Kahn reaction was present in one. In no instance was evidence of luetic heart disease found at necropsy. Arteriosclerotic changes were found at necropsy in all the cases of this series and peripheral arteriosclerosis was evident in practically all the patients.

Associated Arrhythmias.—The striking feature in this relatively small series is the number of electrocardiograms showing arrhythmias other than premature contractions, the latter being the type most frequently reported in the literature. Of the eighteen cases five showed auricular fibrillation, although it was difficult to determine in two of these the duration of the irregularity. One of these, Case 14, showed short periods of ventricular tachycardia following symptoms of coronary occlusion which occurred while the patient was in the hospital. In Case 3 persistent ventricular tachycardia superseded the auricular fibrillation which occurred following the thrombosis and was apparently the immediate cause of the patient's death. Although ventricular tachycardia occurred four times in this series, in only two of the patients did it follow immediately the attack, and in one of these (Case 13) it was controlled by quinidin, the patient dying suddenly several days later while the rhythm was regular. Complete heart block was encountered twice, and in each case it apparently occurred as a direct result of the coronary accident. The remaining nine cases showed regular rhythm with the exception of occasional ventricular premature contractions in three of these. Bundle branch block involving the right bundle occurred once.

Electrocardiograms. — The electrocardiograms in six patients of this series failed to show changes in the R-T interval which are considered typical of coronary occlusion. The so-called

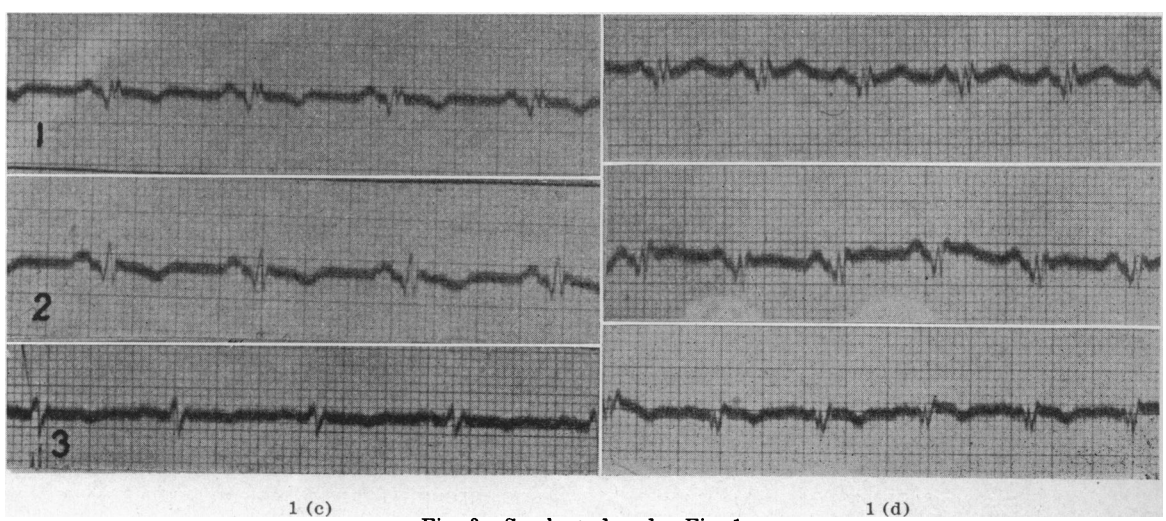

Fig. 2.—See legend under Fig. 1.

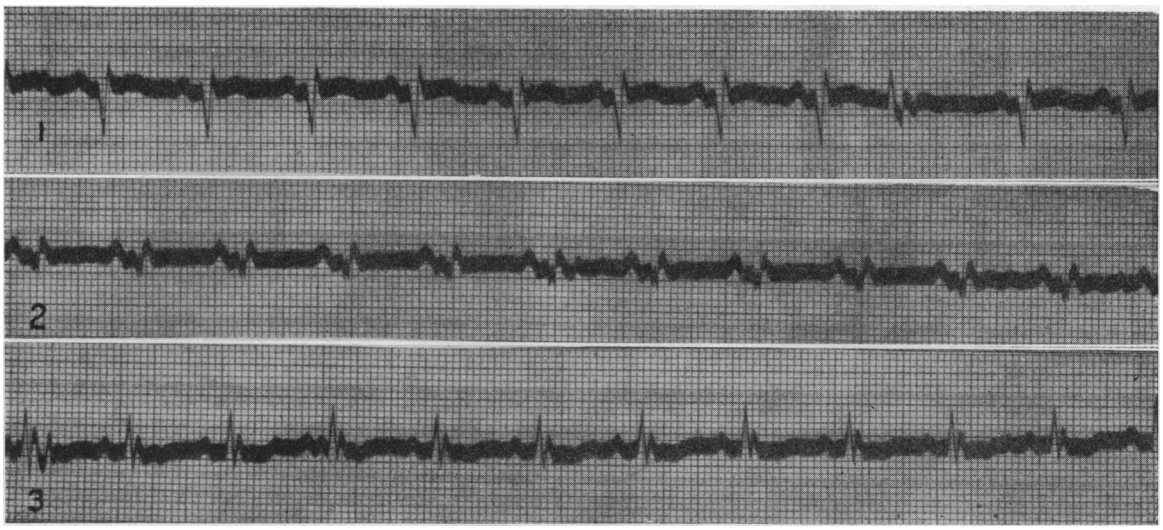

Fig. 3.—Electrocardiogram taken March 5, 1931, showing right axis deviation, ventricular premature contraction low voltage and slight upward convexity of the R-T interval in lead 1.

"coronary T" occurred in lead 1 alone, four times; and in leads 1 and 2, twice; in leads 2 and 3, three times; lead 3, two times; and once in all three leads. The patients in whom no suggestive changes in the R-T interval were detected were as follows: three with regular rhythm, two with auricular fibrillation, and one with complete heart block.

Coronary Involvement.—The infrequency with which occlusion of the right coronary artery is encountered at necropsy is demonstrated in this study. Only two patients showed complete occlusion of the right, although in two there were marked sclerosis and narrowing in addition to thrombosis of a branch of the left coronary artery. In one of these patients the electrocardiogram showed auricular fibrillation with typical R-T changes later followed by ventricular tachycardia, while the other showed complete heart block with no R-T interval changes. In fifteen cases the left coronary was involved and in one the pathologist failed to state the location of the thrombus. No attempt was made to localize the lesion by means of the electrocardiogram, but the impression remains after analyzing these case histories that this would not have been possible.

REPORT OF CASES

Lack of space prevents a detailed report of all the cases in this series. Two cases, No. 5 and No. 13, have been selected for description.

Case 5.—A man, fifty-four years old, was admitted March 31, 1927, complaining of pain beneath the sternum and in the back and shoulders. The onset occurred five days previously with sharp, lancinating pain in the precordial area and under the left scapula. These symptoms were accompanied by shortness of breath and cyanosis. Except for frequent colds and sore throats, and an attack of rheumatism ten years previously, the patient denied all other illnesses including venereal disease. He stated that he used neither tobacco, alcohol, nor drugs. On admission the patient was dyspneic and cyanotic. The heart rate was rapid and the rhythm was of the "gallop" type. The heart sounds were of poor quality, but no murmurs or friction rubs were heard. There was an occasional premature contraction. The blood pressure was 106 systolic and 80 diastolic. An electrocardiogram taken shortly after admission showed low voltage in all leads, and a high take-off of the R-T interval in leads 2 and 3. The record was considered typical of coronary occlusion. Four subsequent electrocardiograms during the next five months showed a persistence of the low voltage, but the high take-off of the R-T interval became less noticeable (Figs. 1 and 2).

The patient's course in the hospital was very stormy during the first three months. He suffered frequent attacks of severe precordial pain with radiation to the left arm, and at times severe nausea was present. The blood pressure gradually rose from 106 systolic and 80 diastolic to 165 systolic and 100 diastolic. Five months after admission the patient developed symptoms which were attributed to either bronchopneumonia or infarction in the right lung. The leukocyte count rose to 31,000 with 88 per cent polymorphonuclears. The temperature remained elevated for a week, following which definite improvement was noted. A small amount of sterile hemorrhagic fluid was removed from the right thorax during this acute illness. The patient was discharged September 28, 1927. Treatment consisted of rest, nitrites, theocalcin, and sedatives.

Following the patient's discharge nothing further was heard from him until December 1929, when he was sent to the out-patient department by another clinic for an orthodiagram. This showed a slight decrease in the size of the heart. He was again lost sight of until March 5, 1931, when he was admitted to the medical service with a diagnosis of cardiac decompensation. He had been taking small quantities of digitalis over a long period of time, but one month prior to this admission he had developed increased

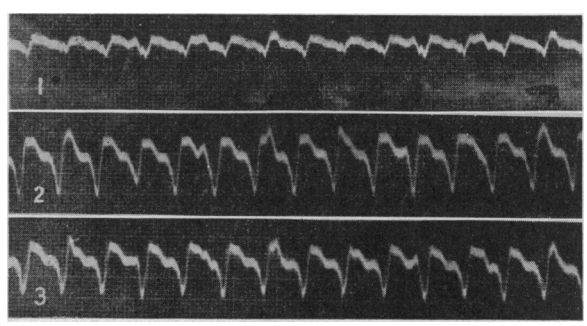

Fig. 4.—Electrocardiogram of J. K., showing ventricular tachycardia with a rate of 150.

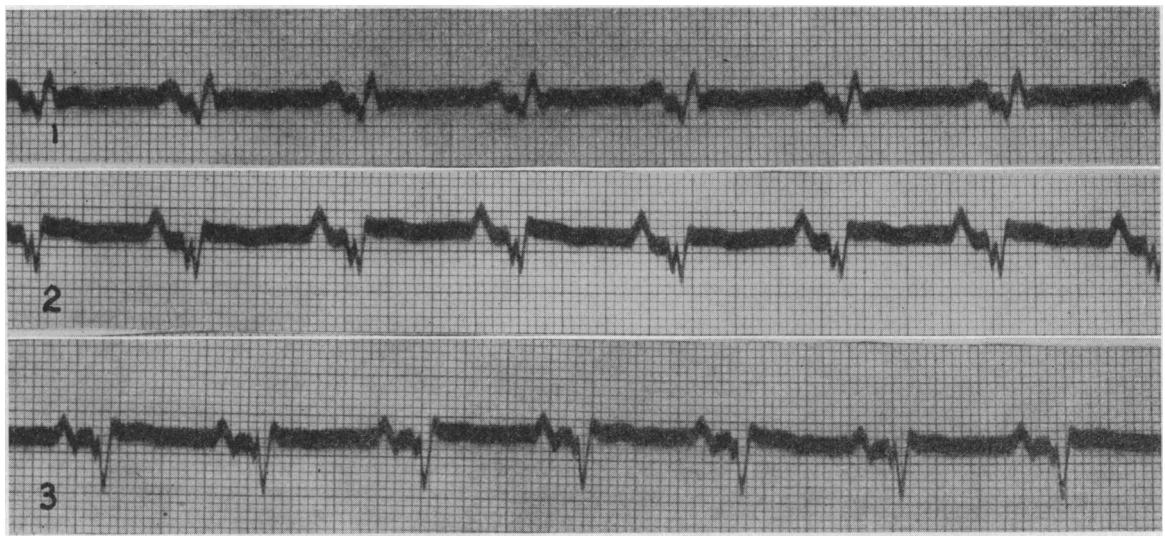

Fig. 5.—Electrocardiogram of J. K. after two grams of quinidin sulphate by mouth had been administered (0.3 gram every three hours), showing normal rhythm.

dyspnea and weakness, and edema had appeared in the lower extremities. The patient stated that he had been able to do light work since his discharge from the hospital in 1927, but he had experienced frequent attacks of weakness, palpitation, and dyspnea. There had been little or no pain. Examination revealed the general picture of congestive heart failure. There was marked arteriosclerosis. The blood pressure was 120 systolic and 80 diastolic. The heart rate was 90 and the rhythm was regular. An electrocardiogram taken five days after admission showed right axis deviation, ventricular premature contractions, low voltage, and a slight upward convexity of the R-T interval in lead 1 (Fig. 3). The patient was given digitalis and the fluid intake was restricted. He died suddenly twelve days after admission while sitting up in bed.

At necropsy the heart weighed 500 grams. All chambers were markedly dilated and there was slight hypertrophy of the ventricles. On the anterior surface of the endocardium of the left ventricle there was an area 3 x 4 centimeters covered with material resembling a thrombus. On section the greater part of the wall of the left ventricle, especially that portion overlying the thrombotic area, was very fibrous and contained numerous petechiae. The anterior descending branch of the left coronary artery showed many calcareous plaques with marked narrowing of the lumen and obliteration of the artery about one centimeter from its origin. Similar changes were observed in the circumflex branch of the left coronary with almost complete obliteration of the lumen near its termination.

Electrocardiograms taken following the acute onset in 1927 were considered characteristic of coronary occlusion, but the record obtained during the patient's last illness showed only low voltage, occasional ventricular premature contractions and a slight upward convexity of the R-T interval in lead 1. In the earlier electrocardiograms the characteristic R-T changes were found in leads 2 and 3. Necropsy disclosed no evidence of recent thrombosis, but the findings revealed obliteration of the lumen of the anterior descending branch of the left coronary artery and a fibrosis of the wall of the left ventricle.

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Case 13.—A man, fifty-one years of age, was admitted to the hospital complaining of marked weakness, dyspnea, palpitation, nausea, and vomiting. The symptoms had first occurred eight days previously, but had been much worse for two days prior to admission. There had been no previous similar attacks. There had been considerable precordial pain. On admission the blood pressure was 98 systolic and 80 diastolic. The heart rate was extremely rapid, the rhythm was regular and the sounds were practically inaudible. An electrocardiogram obtained shortly after admission showed ventricular tachycardia with a rate of 150 (Fig. 4). The patient was given quinidin sulphate by mouth, 0.3 gram every three hours, and after 2 grams had been administered the rhythm was normal (Fig. 5). The patient appeared to be improving, but he died suddenly while sitting up in bed six days after admission.

At necropsy the heart weighed 500 grams. There was marked coronary sclerosis with complete occlusion by a thrombus of the interventricular branch of the left coronary artery. There was fibrosis of the myocardium at the apex.

The electrocardiogram taken eight days after the onset showed ventricular tachycardia. Sinus rhythm was restored by the oral administration of quinidin.

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DISCUSSION

Donald J. Frick, M. D. (1136 West Sixth Street, Los Angeles).—The cases reported have been well

analyzed and, fortunately, leave no question in our mind as to the diagnosis as they were followed to the autopsy table. All showed marked sclerosis with probable cardiac fibrosis, so that it is not surprising that there was such a large number evidencing abnormal rhythm during some stage of their illness. Auricular fibrillation has not been an unusual finding in our cases of coronary occlusion in the aged, and occurred in one young man (whose usual health was robust) who developed a coronary occlusion following an operation for a gangrenous appendix. Heart block, bundle branch block, may not be a sequel of coronary occlusion but may have existed for some time prior to the occlusion. The same mechanism that causes coronary occlusion oftentimes produces fibrotic changes in the conducting system with consequent block. We have wondered at times whether the change in the circulation of the heart was the cause of abnormal rhythm, as we have seen several patients develop auricular fibrillation of a paroxysmal type following sudden shock or injury: one patient after being badly frightened, one following a fractured clavicle, and two after fracture of the hip. All these patients had exhibited myocardial damage of an appreciable degree with hypertension and arteriosclerosis but no evidence of coronary occlusion at the time of the onset of their abnormal rhythm.

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WILLIAM DOCK, M. D. (Stanford University Medical School, San Francisco).—Doctor Leake's carefully studied series of patients who died following coronary occlusion emphasizes the serious import of paroxysmal tachycardia in this condition, for the incidence is always, as in his series, greater in those who are going to die of the disease than in those who survive an attack of this sort. On an experimental basis, both digitalis and quinidin have been shown capable of preventing such attacks of tachycardia, but it is the usual practice to use quinidin. It is now quite clear that patients who have had extreme shock and severe myocardial damage due to coronary thrombosis tolerate quinidin even in very large doses, and I know of no cases in which its use has done harm. The need for it usually arises between the third and fourteenth days after the onset of symptoms. Heart block, either transient or permanent, is likely to manifest itself earlier, but is a less serious complication and requires treatment only when associated with Stokes-Adams seizures.

I believe that the safest administration of quinidin is to begin the drug in doses of 0.3 to 0.5 gram every four to six hours as soon as paroxysmal tachycardia occurs or when ventricular extrasystoles become more frequent than six to eight per minute, and to continue the drug for at least two weeks, regardless of whether the arrhythmia is abolished quickly or not. I should be interested in knowing if Case 13 was still on quinidin at the time of his death. Quinidin, unlike digitalis, has a rather transient effect, and must be given every six hours to keep the heart under its influence. While we cannot expect it to prevent sudden death in this group of cases, it undoubtedly reduces the risk of ventricular fibrillation in the patients with ventricular arrhythmias.

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V. R. MASON, M. D. (838 Pacific Mutual Building, Los Angeles).—This timely paper by Doctor Leake calls attention to sudden disturbances of rhythm in patients and to their importance as evidence of disease of the coronary arteries.

It is the demonstration of these minor disturbances as well as the more spectacular phenomena which has led to more early and accurate diagnosis of cardiac disease incident to coronary artery narrowing.

With early and accurate diagnosis it is apparent that the institution of proper treatment will lead to considerable prolongation of life in many instances.

THE PATCH TEST—ITS USE IN DERMATOLOGY*

REPORT OF CASES

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DISCUSSION *by Fred Firestone, M. D., San Francisco; Norman N. Epstein, M. D., San Francisco; Samuel Ayres, Jr., M. D., Los Angeles.*

THE patch test was suggested in Europe by Bloch and Jadosshon. Interest has been stimulated in this country particularly by Sulzberger, Wise, Spain, and others.

This test is of assistance in determining the etiology of many cases of dermatitis venenata. Of recent years many attempts have been made to clarify the subject of dermatitis and eczema. Investigation has succeeded in amputating various groups from the old classification. A better understanding of the part played by fungi in the production of dermatoses has removed a large number of conditions from the old eczema group and has established them as distinct entities. With the patch test we are now in the process of segregating another group.

The test itself will probably have a wide use. It is simple to apply, does not require much time, and the results are frequently easily apparent.

TECHNIQUE

The test is performed as follows: The gummed surface of a strip of adhesive tape is partly covered with a piece of oiled silk about one inch square. Gauze or linen, two or three layers thick, is cut about one-half inch square. This gauze is moistened with the suspected material. The gauze square is placed in contact with the skin and covered by the oiled silk and adhesive tape. Spain has used ordinary blotting paper instead of gauze and found it satisfactory. If the tests are few they may be applied to the arm. Frequently, however, there are many suspects so that it is necessary to apply the tests to the back. Ordinarily the patches are left in contact with the skin twenty-four hours. If, however, a severe reaction results, the patch should be removed. Spain, in his work of poison ivy, found that contact for two to four hours was sufficient.

It is now a well-established opinion that many cases of dermatitis or eczema of an erythematous, papular and vesicular type are truly allergic phenomena. Much of the experimental work has been done with plants such as primrose. Bloch, for instance, has succeeded in sensitizing himself to primrose. There are so many individuals who are sensitive to plants that experiments can be repeated sufficiently often to be convincing.

It is well known that in certain cases of urticaria, the patient can be shown to be sensitive to various substances. This has been demonstrated by scratch and intradermal tests. In eczema, how-

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